

**A model of motor neuron loss: selective deficits after ricin injection.**

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**Public Summary:**  
None

**Scientific Abstract:**

This study characterizes a model of motor neuron (MN) loss on the molecular, cellular, and behavioral levels. Injection of the toxic lectin Ricinus communis agglutinin I (RCA I or ricin) caused cellular deficit and loss of function by damaging the sciatic nerve. Since the sciatic nerve supplies movement to most of the lower limb, damaging this motor system models lower limb paralysis and the deficits that occur in diseases like amyotrophic lateral sclerosis (ALS) and infantile progressive spinal muscular atrophy (SMA). We used motor-, sensorimotor-, locomotor-, and reflex-based tests to demonstrate loss of function after ricin injection. Loss of function was also demonstrated by decreased retrograde transport, and supported by measurements of muscle wasting. Histochemical and molecular methods were used to characterize sciatic nerve damage in axons and cell bodies, including apoptotic cell death in MNs. This battery of tests documents the extent of the ricin-induced damage and provides a baseline that can be used to judge the efficacy of MN treatment strategies in preclinical studies.

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